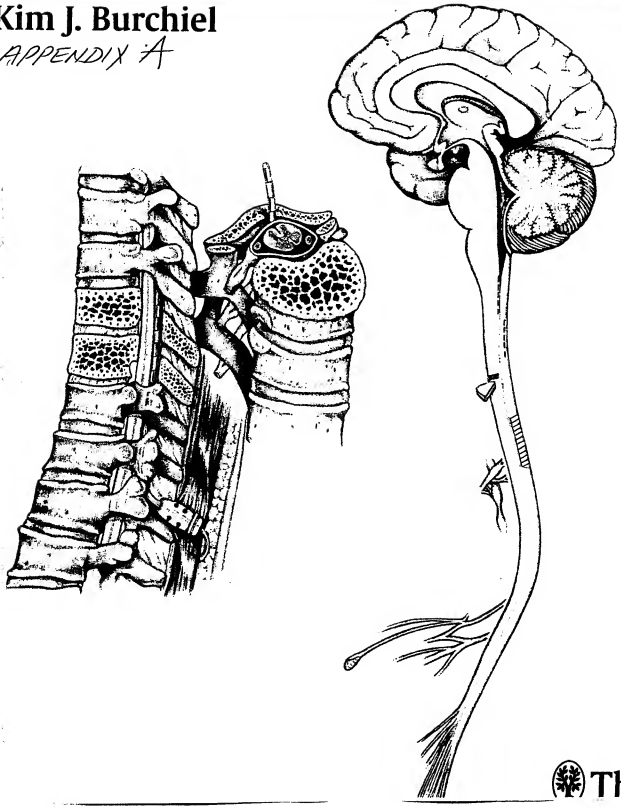
Surgical Management of Pain

Kim J. Burchiel

APPENDIX A



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Appendix

Chapter 44

Deep Brain Stimulation for Chronic Pain

Ali R. Rezai and Andres M. Lozano

Recent advances in anatomic and functional imaging, improvements in physiological localization techniques, wide availability of stereotactic systems, and the realization of the limitations of medical therapy have resulted in a renaissance in the use of deep brain stimulation (DBS). The chronic electric stimulation of deep (subcortical) brain targets, or DBS, is becoming an increasingly used mode of therapy in stereotactic and functional neurosurgery. The major advantage of DBS in contrast to traditional lesioning procedures is its adjustable and reversible feature that allows maximal clinical efficacy while minimizing complications. Currently, the most common application of DBS is in movement-disorder surgery. The improved safety and the striking benefits of DBS have expanded the possibilities of intervention into novel targets, including the subthalamic nucleus (STN) and the globus pallidus.[1-7] With the technical and scientific advances that have taken place in recent years, it is now appropriate to reexamine the use of DBS for the treatment of pain.

The exact mechanism of DBS action is unknown. Prevailing theories implicate a depolarizing blockade or "jamming" of neurons and an activation of axonal tracts. The effects of high-frequency (>100 Hz) stimulation in areas populated by neuronal cell bodies often mimic those seen with lesioning the same structure. The same parameters of stimulation in axonal projections such as the corticospinal or the optic tracts, however, appear to activate these projections. Both anterograde and retrograde effects occurring locally or having far-reaching transynaptic effects are possible. Whereas high-frequency stimulation (>100 Hz) is often necessary and is routinely used in movement disorder surgery, lower-frequency stimulation has been efficacious in pain surgery. The reasons for this are unknown at this time.

Although DBS for chronic pain had been used for many years, it had fallen out of favor over the past decade, particularly in the United States. This is partially attributed to improvements in oral and intrathecal therapy for pain, particularly the widespread use of opioid therapeutic regimens such as intrathecal pumps, combined with the perceived poor outcomes reported in the literature. This chapter provides an overview of the history of DBS, its proposed mechanism of action, current indications, clinical applications, and future prospects.

HISTORICAL PERSPECTIVE

Based on the observation of positive reinforcement and "pleasure centers" identified with brain stimulation in rodents,[8] neurosurgeons ventured into the field of electric stimulation of the brain for the relief of pain. Early efforts using temporarily implanted electrodes eventually were replaced by chronic brain stimulation through permanently implanted electrodes coupled to battery-powered pulse generators.

Deep brain stimulation for chronic pain has been used since the 1950s, when Heath[9] and Pool[10] implanted temporary electrodes in the septal region for psychosurgery in patients with schizophrenia and metastatic carcinoma. The electrodes were placed in the septum pellucidum in a region anterior and inferior to the foramen of Monro with successful pain relief. Pool et al in 1956[11] and Heath and Mickle in 1960[12] reported pain relief with septal stimulation in nonpsychiatric chronic pain patients. Ervin et al[13] reported analgesia induced by stimulation of the caudate nucleus in a patient with intractable facial pain secondary to carcinoma of the pharynx and skull base. The pain relief lasted at times for 6 to 8 hours after the stimulation was turned off. Gol[14] stimulated both the caudate nucleus and the septal region in six patients with intractable pain, but successful pain relief was obtained in only one patient.

Despite these earlier reports of septal and caudate stimulation, the current applications of DBS for pain involves either paresthesia producing thalamic, medial lemniscus, or internal capsule stimulation or periventricular gray (PVG)/periaqueductal gray (PAG) stimulation.

The concept of thalamic stimulation arose from the Melzack–Wall gate theory of pain,[15] that stimulation of large myelinated fibers of the peripheral nerves resulting in paresthesias would block the activity in small nociceptive

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projections. Thalamic stimulation for pain relief was envisioned within the spectrum of modulation of sensory and pain pathways in the neuraxis at the level of the spinal cord via dorsal column stimulation and more rostrally to the lemniscal pathways, thalamic sensory relay nuclei [ventralis caudalis (VC)/ventralis posterior (VP)], and sensory portion of the internal capsule.[16]

Thalamic stimulation for pain relief was first reported by Mazars et al,[17] who reported paresthesia producing stimulation along the ventroposterolateral (VPL) nucleus relieved chronic intractable deafferentation pain. Hosobuchi et al[18] reported the efficacy of chronic ventroposteromedial (VPM) stimulation using 100-Hz frequency in patients with refractory facial pain. Subsequently, Hosobuchi and Adams[18] and Mazars et al[19] reported long-term success using chronic implantable VP stimulators in patients with deafferentation pain.

Another region where stimulation evoked paresthesias was the internal capsule. Fields and Adams observed that stimulation in the region of the internal capsule provided pain relief.[20] Subsequently Adams[16] and Fields and Adams[20] implanted chronic stimulating electrodes in the posterior limb of the internal capsule in a number of patients with pain relief. Cooper et al[21] also reported similar efficacy, with internal capsule stimulation resulting in pain reduction in a patient with lower-extremity pain and spasticity following spinal cord injury.

During the same time that paresthesia-producing stimulation was being actively pursued, animal studies of Reynolds[22] demonstrated that stimulation of the lateral margin of the PAG in rats induced analgesia, enabling abdominal surgeries to be performed on awake animals without the use of anesthetics. Confirmation of the efficacy of this method in humans, using frequencies ranging from 10 to 75 Hz and pulse widths of 200 to 250 msec, was reported in 1977 by Richardson and Akil.[23,24] Hosobuchi et al[25] reported similar findings also occurring with PVG stimulation. Furthermore, the stimulation effects were reversible with the opiate antagonist naloxone, indicating that the mechanism of pain relief was opioid mediated. Biovie and Meyerson[26] and Young and Rinaldi[27] also showed that stimulation may involve the dorsal medial nucleus (DM) and the parafascicular nucleus (Pf).

Although DBS involves stimulation of subcortical structures, it should also be recognized that there have been several studies supporting the role of motor cortex stimulation for pain control. In the process of performing sensory cortex stimulation in an attempt to relieve thalamic pain, Tsubokawa and colleagues found that stimulation of the precentral gyrus/motor cortex was effective in relieving thalamic pain.[28,29] Interestingly, stimulation of the sensory cortex exacerbated the pain in many patients. Subsequently, a number of other investigators, including Meyerson et al[30] and Katayama[31] reported promising results with refractory trigeminal neuropathic pain and other types of central pain. Additional studies are warranted to elucidate more fully the role of motor cortex stimulation for chronic pain management. These studies provide support for the notion that the term *DBS* may need to be changed to the more generic term *intracranial neural stimulation.*

GENERAL CONSIDERATIONS

Pain Characteristics

Chronic pain can be generally characterized as being nociceptive or nonnociceptive (Table 44–1). *Nociceptive* or *somatic pain* is caused by direct activation of the nociceptors (mechanical, chemical, and thermal) found in various tissues. The afferent somatosensory pathways are intact in nociceptive pain. Examples include cancer pain from bone or tissue invasion or noncancer pain secondary to degenerative bone and joint disease or osteoarthritis.

Nonnociceptive pain, occurring in the absence of activation of peripheral nociceptors, also has been referred to as *neuropathic pain* or *deafferentation pain.* This type of pain results from an injury or dysfunction of the central or peripheral nervous system. This damage can occur anywhere along the neuraxis. Examples include thalamic pain, stroke, traumatic or iatrogenic brain or spinal cord injuries, phantom limb or stump pain, postherpetic neuralgia, and various peripheral neuropathies.

Patient Selection

The initial choice for treating the chronic pain patient is a conservative approach involving medications (narcotic and nonnarcotic analgesics, antidepressants, anticonvulsants), physical therapy, biofeedback, transcutaneous electrical nerve stimulation (TENS), as well as less conventional or alternative therapies. Once conservative approaches have been exhausted, more invasive procedures can be considered: blocks, neurolysis, or other ablative procedures. Neu-

TABLE 44–1 Characteristics of Neuropathic and Nociceptive Pain

Nociceptive pain
Pain secondary to activation of nociceptors
Somatosensory afferent pathways are intact
Most commonly described as sharp, dull, throbbing pain
Responds to narcotic analgesics
Responds to ablative/lesioning procedures
Neuropathic/deafferentation pain
Pain secondary to a lesion, injury, or dysfunction of the CNS/PNS
Also referred to as deafferentation pain
Most commonly described as constant, steady, burning, aching, dysesthetic pain
Also neuralgic (sharp, shooting) and evoked (allodynia, hyperpathia) components
Less responsive to narcotic analgesics
Responsive to narcotic analgesics
Responsive to antidepressants
Not responsive to ablative/lesioning procedures

CNS, central nervous system; PNS, peripheral nervous system.

roaugmentative techniques such as spinal cord stimulation or intrathecal pumps also may be beneficial. With failure of these measures and persistent incapacitating pain, the patient becomes a candidate for brain stimulation.

Prior to proceeding with DBS, patients must satisfy a general selection criteria (Table 44–2) and be treated by a multidisciplinary pain management team. Additionally, a minimum of 6 months should have passed after pain onset prior to consideration for brain stimulation. We generally reserve DBS for patients who have pain they regard as severe and incapacitating and that is 6 or greater (of a maximum of 10) in intensity on a visual analog pain scale.

The patients must have persistent, severe, incapacitating pain despite exhausting all previous less invasive treatment modalities. The pain should be the predominant problem causing disability and suffering for which the patient seeks relief. Patients with long-standing pain complaints without a clearly defined etiology are not candidates.

SPECIAL CONSIDERATION

DBS for chronic pain should be used only in patients who are incapacitated and have failed to respond to all other therapeutic modalities.

Psychiatric evaluation by an experienced team is crucial to exclude patients with significant psychological or psychosocial overlay or secondary gain. In general, patients with psychosis or a strong psychopathology should be encouraged to undergo further psychological treatment. It should be recognized, however, that most patients with refractory chronic pain have mild psychological disturbances, such as depression and anxiety. Specific psychological tests such as the Minnesota Multiphasic Personality Inventory[32] can be helpful in the selection process; patients with high hypochondriasis and hysteria indices have been associated with a poorer outcome post surgery.[33]

Table 44–3 lists various chronic neuropathic and nociceptive conditions that have been treated by using DBS. The choice of stimulation site is determined by the pathophysiology of the patient's pain. In general, patients with refractory neuropathic pain should undergo paresthesia-producing stimulation, whereas those with nociceptive pain should undergo PVG/PAG stimulation. In reality, most pain syndromes have mixed components of nociceptive and neuropathic pain; thus, both a paresthesia-evoking and a PVG/PAG stimulation trial is performed. In the context of neuropathic pain, the steady component responds best to paresthesia-producing stimulation; the evoked (allodynia and hyperpathia) and neuralgic elements of neuropathic pain also may be helped by PVG/PAG stimulation. Overall, DBS is an option for patients with chronic refractory and incapacitating nociceptive and neuropathic pain.

SPECIAL CONSIDERATION

The evaluation of the patients by a multidisciplinary pain center and strict adherence to the selection criteria are crucial for the optimal response because a significant percentage of failures can be attributed to improper patient selection.

TABLE 44–2 Deep Brain Stimulation for Chronic Pain: General Selection Criteria

Evaluation and treatment by a multidisciplinary pain team
Persistent, severe, incapacitating pain
Failure of all previous treatment modalities
Pain is the predominant complaint
Absence of major psychological or psychosocial overlay
Clear understanding that
Procedure is not curative
50% reduction in pain is an anticipated result
Pain recurrence can be common

MECHANISMS OF PAIN MODULATION

Paresthesia-Producing Stimulation

The exact mechanism by which paresthesia-evoking thalamic stimulation results in pain relief is not known, but it is most likely a nonopioid mechanism. One concept is that deafferentation causes an abnormal firing pattern in thalamic neurons and that thalamic stimulation inhibits this abnormal neural activity. Gerhart et al[34] showed that stimulation of the VPL, the primary somatosensory relay nucleus, in monkeys caused inhibition of spinothalamic neurons' evoked responses to noxious cutaneous stimulation. Thalamic stimulation caused a greater reduction of the response to C-fiber volleys than A-fiber volleys.

In studies of chronic pain patients, an abnormal pattern of neuronal firing has been shown in the sensory thalamus in

TABLE 44–3 Chronic Pain Conditions Treated with Deep Brain Stimulation

Neuropathic/deafferentation	Nociceptive
Anesthesia dolorosa	Failed back
Iatrogenic trigeminal pain	Osteoarthritis
Stroke	Cancer pain
Thalamic syndromes	
Brachial plexus avulsion	
Postherpetic neuralgia	
Postcordotomy dysesthesia	
Spinal cord injuries	
Peripheral neuropathies	

patients with central deafferentation pain.[35] Lenz et al[36] showed that areas in the somatosensory thalamus that had lost their normal innervation had abnormal spontaneous bursting activity, with electric stimulation inducing burning dysesthesias. Similarly, Rinaldi et al[37] and Lis-Planells et al[38] demonstrated an increased number of bursting neurons in the medial and lateral thalamic nuclei in deafferented rats compared with controls, along with an increase in the number of nociceptive responsive neurons in the medial thalamus.

Periventricular/Periaqueductal Stimulation

The mechanism of pain modulation with PVG/PAG stimulation is most likely via an opioid dependent pathway. Elevations of endogenous opioids, such as beta endorphin and metenkephalin, have been demonstrated in cerebrospinal (CSF) samples from the third ventricle after PVG or PAG stimulation but not with VC stimulation.[39,40] Several intralaminar thalamic nuclei, including the centrum medianum (CM) with Pf and centralis lateralis (CL), also may contribute to pain modulation.[41,42]

The neural substrates of the endogenous analgesia pathway include the PAG, the nucleus raphe magnus (NRM), the locus coeruleus (LC), and the magnocellular part of the nucleus reticularis gigantocellularis (Rmc).[43] These pathways involve descending projections from the midbrain to the dorsal horn as well as to various thalamic intralaminar and medial nuclei. Further evidence for an ascending as well as a descending endogenous opioid pathway was provided by Peschanski and Besson,[44] who demonstrated projections form the NRM to the intralaminar nuclei and the nucleus submedius. Also, stimulation of the dorsal NRM inhibits the responses to noxious stimuli of Pf neurons.[45]

OPERATIVE TECHNIQUE AND PHYSIOLOGIC MAPPING

The initial steps in stereotactic procedures are similar. The general stereotactic technique is described first. Specific details pertaining to targeting and stimulation are discussed in subsequent sections.

Head-Frame Application and Stereotactic Imaging

Patients undergo application of the stereotactic head frame under local anesthesia. At our institution, we use the Leksell model G frame (Elekta Instruments, Atlanta, GA, U.S.A.); however, any of the commercially available stereotactic systems can be used. After frame application, patients undergo either computed tomography (CT) or magnetic resonance imaging (MRI). It is not necessary to perform ventriculography for these procedures because the currently available MRIs with high-resolution volumetric acquisitions obviate the need for this invasive procedure. MRI has higher anatomic resolution than CT, but it is more susceptible to distortions in spatial accuracy. Any of the currently used image-correction algorithms or MRI–CT image fusion can minimize these distortions.

At the Toronto Hospital, a Signa 1.5-tesla magnet (General Electric, Milwaukee, WI, U.S.A.) is used. After the initial acquisition, a midline sagittal slice is chosen to identify the anterior commissure (AC) and posterior commissure (PC). A subsequent volumetric scan is performed along the AC–PC line, resulting in coronal, sagittal, and axial sections. Images are acquired using a gradient echo sequence with a relaxation time of 43 msec and an excitation time of 13 msec (flip angle 450, FOV 24 × 24, matrix 256 × 256). The sections are 1 mm thick and nonoverlapping.

Anatomic Target Localization

Anatomic localization of the thalamic sensory nucleus VC, medial lemniscus (ML), internal capsule (IC), PVG, or PAG can be achieved in two ways: (1) by targeting using measurement in relation to the AC and PC and (2) indirectly using a standardized anatomic brain atlas as a function of the AC and PC.

The indirect targeting approach uses the stereotactic AC and PC coordinates that are entered into a laptop or desktop computer with a commercially available program containing digitized diagrams of sagittal brain sections from a standardized brain atlas.[46] The program transcribes the patient's calculated AC–PC intercommissural line onto the digitized map at the sagittal laterality of interest. On these maps, structures such as the VC and ML can be localized. The subsequently generated brain map is overlaid onto a millimeter grid ruled in stereotactic coordinates in the anteroposterior and dorsoventral scales with a corresponding diagram of the brain nuclei and tracts depicted in the chosen laterality (Fig. 44–1). The laterality of the maps is chosen according to the location of the pain. Typical laterality is 12 to 14 mm from the midline for facial pain, 14 to 15 mm for upper-extremity pain, and 15 to 17 mm for lower-extremity pain.

Surgical Procedure

Subsequent to the stereotactic CT/MRI acquisition and anatomic target localization, the patient is taken to the operating room. The procedure is performed with the patient under local anesthesia. A linear or curvilinear incision is made 2.5 cm lateral to the midline. A 14-mm burr hole is made anterior to the coronal suture. The dura is opened, and fibrin glue is applied to minimize CSF leaks and entry of air into the cranial cavity. The stereotactic arc then is applied, and the X, Y, and Z coordinates for the anatomical target area are set. A guide tube cannula with a blunt-tip stylet then is introduced into the brain parenchyma to a point 10 mm proximal to the chosen target. At this time, physiological localization starts, with the ultimate aim of correlating the anatomic and physiologic findings.

Physiologic Target Localization

Sole reliance on anatomic localization can be problematic because of the frequent discrepancy between the expected location and actual position of the stereotactic targets. Physiologic corroboration can be achieved with microelectrode recording and stimulation or macrostimulation. Macroelectrode stimulation is rapid, it requires minimal equipment,

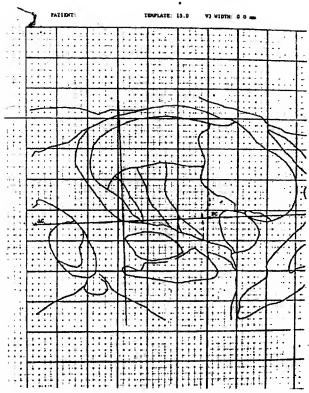

Figure 44–1 Anatomical brain map. Computer-generated sagittal brain diagram 14 mm from the midline (using digitized Schaltenbrand and Wahren atlas), drawn with the AC–PC intercommissural distance adjusted to that of the patient and overlaid onto a grid (mm) ruled in stereotactic coordinates in the anteroposterior and dorsoventral scale of the frame applied to the patient's head.

SPECIAL CONSIDERATION

Physiologic localization is mandatory for definitive target determination in DBS for pain.

but it has low spatial resolution, not being able to record neurons, and it is unable to discriminate between axons and neurons. Microelectrodes, however, provide exquisite physiologic identification of receptive fields and neuronal firing patterns through direct measures of individual single unit neuronal activity and are able to distinguish somatodendritic from axonal activity. The characteristic physiologic mapping findings of microelectrode recording and microstimulation or macrostimulation for specific target are discussed in the following section.

Paresthesia-Evoking Targets

The paresthesia-producing targets are the thalamic sensory relay nucleus (VC–ventralis caudalis), the ML, and the IC. The target is typically 12 to 14 mm from midline for the stimulation of the face, 14 to 15 mm for the upper limb, and 15 to 17 mm for the lower limb. The ML can be targeted inferior to the intercommissural line 12 to 14 mm from the midline. Electrodes are generally placed contralateral to the area of pain. In patients with bilateral pain, electrodes are placed on both sides, usually in single or staged procedures. For focal neuropathic pain, the somatotopic representation of the patient's pain area in the sensory nucleus (VC) is targeted. For more diffuse or hemibody pain, the ML or the IC is chosen to obtain more widespread coverage. With pain resulting from destructive thalamic lesions, microelectrode mapping of the thalamus may be of poor yield,[47] and thus the thalamic afferent or efferent projections (i.e., internal capsule, medial lemniscus) can be targeted for stimulation.[18]

Microelectrode recording in the VC reveals a somatotopic representation of body parts expressed with discrete tactile receptive fields. With microstimulation, patients experience somatotopically organized paresthesias, defining a projected field for these thalamic neurons. As an alternative to microstimulation, macrostimulation can be performed every 1 to 2 mm from about 10 mm above to 10 mm below the expected target.

Physiologic mapping in patients with stroke or major deafferentation may vary from the normal observations as a result of neuronal loss, structural anatomic changes, or plasticity. These include an absence of neurons and their corresponding receptive or projected fields, mismatch between receptive and projected fields, somatotopic reorganization, widened or shrunken receptive fields, neuronal bursting activity, and projected fields that evoke burning or pain rather than paresthesias. In situations where physiologic mapping does not provide a clear receptive field map definition, PVG/PAG stimulation can be an alternative option, particularly in patients with the evoked features of neuropathic pain. Alternatively, in cases of stroke or thalamic lesions, stimulation can be performed at a distal site, such as the motor cortex.[28,29]

PAG/PVG Targets

The anatomic targets for the PVG/PAG are typically 2 to 5 mm anterior to the PC, 2 mm lateral to the medial wall of the third ventricle, and at the level of PC.[23,24,48] The microelectrode recordings from the PVG region in humans are not well characterized. PVG stimulation can result in pleasant sensations or warmth and well-being with stimulation frequencies of 25 to 50 Hz. At higher stimulation intensities, PVG stimulation may evoke feelings of diffuse burning or,

SPECIAL CONSIDERATION

Another feature seen with PVG and PAG stimulation is changes in heart rate and blood pressure, which typically are elevated.[51] These cardiovascular changes, however, are not always seen and are seen with higher stimulation thresholds; thus, they should not be relied on routinely.

at times, anxiety. The typical stereotactic coordinates for the PAG are 2 to 3 mm lateral to the midline, just lateral to the aqueduct (1 to 2 mm), at the level of 1 to 2 mm behind the PC, and 2 to 3 mm below the AC–PC line.[49,50] With ventral PAG stimulation, sensations similar to that of PVG are experienced, but dorsal PAG stimulation typically evokes unpleasant sensations of fear, doom, anxiety, and agitation. Additionally, current spread from increased stimulation settings can cause vertical gaze or other gaze abnormalities. In general, we prefer placement of the electrode in the PVG because of the increased adverse effects associated with PAG stimulation.

DBS Electrode Implantation

The physiologic mapping information obtained along each tract or trajectory is annotated on the corresponding stereotactic brain maps, allowing simultaneous visualization and correlation of the physiologic and anatomic findings. At this time, the optimal location for the placement of the DBS electrode is determined. General principles guiding the final implantation involve placement of the electrode at a region allowing for maximal efficacy while minimizing the undesired side effects. The currently used DBS is a quadripolar Medtronic (Minneapolis, MN, U.S.A.) electrode. Each pole or contact is made of cylindrical platinum/iridium alloy and is 1.5 mm long, separated from the other pole by an insulated distance of 1.5 mm or 0.5 mm, depending on the model and preference (Fig. 44–2). The diameter of the electrode is 1.27 mm, and the entire electrode length can be 28 or 40 cm, depending on the model. A specially provided plastic

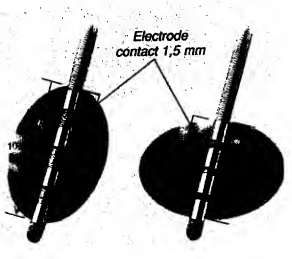

Figure 44–2 The DBS electrode. The deep brain stimulation (DBS) electrode is a quadripolar electrode. Each pole/contact is made of cylindrical platinum/iridium alloy and is 1.5 mm long and 1.27 mm in diameter, separated from the other pole by an insulated distance of 1.5 mm or 0.5 mm, depending on the model and preference (Courtesy of Medtronic Inc., Minneapolis, MN, U.S.A.).

Figure 44–3 Intraoperative target verification. Intraoperative stereotactic radiograph of the DBS electrode at the target. The electrode is introduced from a precoronal burr hole and directed posteriorly and ventrally. The position of the electrode is monitored via fluoroscopy. The cross hairs indicate the target, which is also the center of the stereotactic ring. Note the appearance of the four poles.

ring designed to fit in a 14-mm burr hole is locked into the burr hole.

To confirm the position and trajectory of the actual DBS electrode, intraoperative stereotactic radiography or fluoroscopy is used (Fig. 44–3). Subsequent to insertion of the electrode to the target, a hand-held pulse generator (Screener) is used for intraoperative test stimulation. Various pole combinations and stimulation frequency, pulse width, and intensity are used to determine the thresholds for therapeutic and adverse effects. Thereafter, the DBS lead is locked into the burr hole ring to prevent lead migration, which has been a significant problem in the past. The proximal portion of the DBS lead then is attached to a transcutaneous pacing wire for test trial period. Postoperative MRI imaging confirms the electrode locations (Figs. 44–5 and 44–6).

Test Stimulation Trial Period

Patients undergo test trial period for 3 to 7 days. During this time, various stimulation parameters are used, and a detailed "pain diary" is compiled using visual analog and verbal pain scores. Typical stimulation parameters include unipolar or bipolar stimulation, frequencies of 25 to 75 Hz, pulse widths of 60 to 500 μsec, and variable voltage intensities. A success trial consists of a greater than 50% reduction in the patient's pain with stimulation. If successful, the patient undergoes implantation of a pulse generator or

radiofrequency-coupled receiver. If unsuccessful, the DBS electrode and the transcutaneous wires are removed.

Pulse Generator or Radiofrequency Receiver Implantation

This procedure usually is carried out with the patient under general anesthesia because it involves a tunneling procedure from the frontal incision to the infraclavicular region. The patient is positioned similarly to the position used in a ventriperitoneal shunt. Implantable pulse generator (IPG) device (Itrel III) or RF receiver (Xtel) (Medtronic) are implanted in the subcutaneous tissue in the infraclavicular space. The IPG device is powered by a lithium battery and is fully programmable through telemetry (Fig. 44–4), whereas the radiofrequency device can be activated via an external transmitter using an antenna placed on the skin overlying the receiver.

OUTCOME

In reviewing the literature, it becomes apparent that a carefully designed scientific evaluation of DBS for pain has been a difficult task. Most studies have combined neuropathic and nociceptive pain states along with the combination of different targets. The evaluations for the most part were nonstandardized, and definitions of success differed. Additionally, not all reports distinguished their outcome between those undergoing an initial DBS trial and those who had permanent implants.

In assessing long-term outcomes from previous published studies, the overall results are better for nociceptive pain. In general, PVG stimulation is the most optimal for nociceptive pain and the evoked feature of neuropathic pain, whereas paresthesia-producing stimulation works well for neuropathic pain but not nociceptive pain. Tables 44–4 and 44–5 list some of the reported long-term success

Figure 44–4 The quadripolar DBS electrode and the internal pulse generator. The IPG unit is powered by a lithium battery, which is fully programmable via telemetry

TABLE 44–4 Results of Deep Brain Stimulation for Nociceptive Pain

Series	No. of Patients	Long-Term Success (%)	Follow-up (mo)
Kumar et al, 1997	49	71	84
Levy et al, 1987	57	32	24–168
Hosubuchi, 1986	65	77	24–168
Young et al, 1985	31	81	2–60
Plotkin, 1982	42	81	6–42
Meyerson et al, 1979	76	54	
Richardson and Akil, 1977	20	70	1–46

SPECIAL CONSIDERATION

Studies have shown that the best results from DBS are obtained in patients with cancer pain, failed back pain, cervical brachial avulsions, and peripheral neuropathy. Patients with thalamic pain, postherpetic neuralgias, and traumatic lesions of the spinal cord respond less well.

(generally defined as >50% pain relief) results of DBS for nociceptive and neuropathic pain, respectively.[48,49,52–61] Usually, the success rates decline with time. In general, the best results are in cancer pain, "failed back pain," cervical/brachial avulsions, and peripheral neuropathy. On the opposite end of the spectrum, thalamic pain, postherpetic neuralgia, and traumatic lesions of the spinal cord were among the poorest responders.

COMPLICATIONS

Complications associated with DBS can be categorized as neurologic, technical, and stimulation related.[48,49,52–61] The

TABLE 44–5 Results of Deep Brain Stimulation for Neuropathic/Deafferentation Pain

Series	No. of Patients	Long-term Success (%)	Follow-up (mo)
Kumar et al, 1997	16	44	45
Levy et al, 1987	84	30	14–168
Young et al, 1985	17	59	2–60
Dieckmann and Witzmann, 1982	41	28	6–54
Turnball et al, 1980	18	72	1–47
Meyerson et al, 1979	160	26	